fore, the structure of the abscess-wall would be likely to determine whether an ovarian abscess arose from the Graafian follicle, which is seated partially within the cortex of the ovary, or from the parenchyma of this organ. If the point of infection was a Graafian follicle, the pyogenic wall would contain cells characteristic of that follicle, *i. e.*, luteal cells. If it arose from the parenchyma, there would be an absence of such cells.

In these three abscesses we find the abscess-walls containing these peculiar bodies in the second and third cases, but not in the first one. If, therefore, the premises be correct concerning the histological elements of the abscess-wall in these cases, the deduction that the first case of infection of the ovary must have been through the general circulation, and that of the second and third cases through ruptured Graafian follicles, would seem to be tenable.

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DIPHTHERITIC HEMIPLEGIA.

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Diphtheritic paralyses have been the subject of much research, and much has been written upon them since they were first carefully described, about the middle of the century. Although many things that are peculiar to this form of paralysis remain unexplained, and writers are still divided as to the nature of the lesions, yet since the work of Charcot and Vulpian, who, in 1862,[1] found granular and fatty degenerations of the fibres of the palatine nerves, the more commonly accepted view has been that the process is essentially a peripheral one. Among those who have found lesions of the peripheral nerves besides Charcot and Vulpian are Loraine and Lepine,[1] who also found diseases of the nerves of the palate; Lionville,[2] who found the phrenic nerve affected; Schech,[3] of the recurrent laryngeal nerve; and P. Meyer,[4] who found both parenchymatous and interstitial neuritis; and in some nerves also a neuritis nodosa. Mendel[5] takes the same view, but also found small hemorrhagic infiltrations in various portions of the nervous system.

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[1] Charcot Vulpian: Gazette médicale, 1863.

More or less opposed to these views are those of such men as Dejerine,[6] who holds that the lesions are inflammatory processes, and small hemorrhages in the spinal cord. He found[7] changes also in the cells of the anterior horn of the cord, which he thought comparable to those of anterior poliomyelitis. Although he found proliferation of the nuclei in the sheath of Schwann and drops of myelin, and a loss of the axis-cylinders, he considered these changes secondary. Vulpian[8] also found changes in the cord, but considered them of secondary importance. Kidd[9] ascribes diphtheritic paralyses to changes in the anterior horns of the cord. Buhl[10] found capillary hemorrhages in the brain, in the spinal nerve roots, and in the spinal ganglia, and also calls attention to the changes in the vessels in diphtheria.[11] P. Meyer[12] found, besides the marked peripheral changes extending to the finest end branches of the nerves, also small hemorrhages in the cord.

Oertel[13] found, besides degeneration of the muscles, a fact of importance in connection with thrombosis in the heart in diphtheria, also both large and small hemorrhages. Klebs[14] found small capillary extravasations in the pia of the cerebrum, especially upon the convexity, and occasional greater infiltrations, especially along the course of the sulci, in the ascending frontal convolution, the frontal and the temporal lobes. He also found bacteria, similar to those in the throat, in these hemorrhages. Krauss[15] found normal nerve-nuclei, a degeneration of the peripheral portion of the oculo-motor nerve, and a diapedesis of red corpuscles with greater and smaller hemorrhages throughout the brain. Arnheim[1] found (1) hyperæmia and capillary hemorrhages in the peripheral nerves; (2) slight inflammatory processes in the muscles; (3) severe parenchymatous and interstitial degeneration of the nerve-fibres; (4) no changes in the ganglion-cells; (5) no systemic disease of the cord. Mendel[2] found (1) capillary hemorrhages in the central nervous system, and also in the peripheral nerves; (2) signs of interstitial and parenchymatous neuritis in the form of nuclear proliferation of the neurolemma, and alterations of the medullary sheaths. He says: "Therefore I am inclined to think that the diphtheritic poison attacks the vessel-walls as well as the neurolemma, and indeed at the same time in certain cases, and that the advocates of poliomyelitic, and neuritic changes exclusively, as the basis of the diphtheritic paralyses, may be just as far from the right as those who refer everything to thrombosis and hemorrhages—that is, to changes of the vessel-walls." Recently Bikeles[3] describes small black flakes in the cord, most numerous in the posterior columns (Würzelzone), to which he ascribes the ataxia. Enriquez and Hallion[4] found that the toxins of diphtheria injected under the skin of dogs may cause, if the dose is not too large, intense congestion of the gray matter of the cord, with hemorrhages and foci of inflammation, characterized by formative irritation of the neuroglia. J. J. Putnam[5] says that of the post-infection disorders of the nervous system none are so common as neuritis, and he distinguishes three varieties: (1) The neuritis attending the first stage of an infective process before there has been time for the development of general disorders of nutrition; (2) The post-infectious neuritis, in the production of which general disorders of nutrition and a variety of other causes play an important part; (3) The acute, usually fatal, and hemorrhagic neuritis of unknown origin. "Finally," he says, "it should not be forgotten that what we have considered as neuritis is often associated with nuclear and diffuse spinal lesions, and that better methods of investigation may show these to play a more important part in the production of symptoms and nerve-lesions than has hitherto been supposed." Hochhaus[6] thinks that the cause of diphtheritic paralysis may be situated in the muscles. Besides a slight interstitial neuritis he found always a severe inflammation of the muscles. Cramer[7] thinks that the changes in the nerve are primarily vascular and interstitial. Gowers[1] classifies the paralyses of diphtheria under the form of secondary toxæmic neuritis, in which the toxæmic agent first causes some definite disease, which is followed by neuritis, which is probably generally due to some chemical product of the growth of the organisms in the body. He also says :[2] " Diphtheria may cause acute changes in the nerve-cells and nerve-roots." He also describes the lesions found in the peripheral nerves as primarily parenchymatous, and says :[3] " There is not, as a rule, any inflammatory change in the interstitial tissue of the nerves, and the primitive sheath is generally unchanged." He also speaks of the " parenchymatous and interstitial inflammation and degeneration " of the muscles, and of the frequence with which small hemorrhages have been found throughout the nervous system. Pierret[4] found meningitis in one case, as did also Dejerine and Barth.[5]

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[1] Article on Diphtheria in the Nouveau dictionnaire pratique.

[2] Bulletins de la Société Anat., 1870.

[3] Schech: Laryngologische Mittheilungen. Archiv für klinische Medicin, Bd. xxiii.

[4] P. Meyer: Anatomische Untersuchungen über diphtheritische Lähmungen. Virchow's Archiv., Band lxxxv. p. 181.

[5] Mendel: Zur Lehre von den diphtheritischen Lähmungen. Neurolog. Centralblatt, 1885, p. 128.

[6] Archives de Physiologie normal et pathologique, 1878, p. 107.

[7] Dejerine: Comp. rend., lxxxv. No. 24.

[8] Maladies du système nerveux, 1877.

[9] Lancet, Jan. 13, 1883.

[10] Zeitschrift für Biologie, 1867, p. 341.

[11] Loc. cit., p. 359.

[12] Loc. cit., p. 214.

[13] Ziemmsen: Handbuch, ii. 2te. Auflage, 1876, p. 608 and p. 638.

[14] Eulenburg's Realencyklopädie, 1 Auflage, Bd. iv. p. 167.

[15] Neurolog. Centralblatt, 1888, p. 480.

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[1] Anatomische Untersuchungen über diph. Lähmungen. Archiv für Kinderheilkunde, Bd. xiii.

[2] Loc. cit., p. 133.

[3] Bikeles: Zur pathologischen Anatomie der post-diphtheritischen Lähmung. Arbeiten aus dem Institut für Anatomie und Physiologie des Central-nervensystems an der Wiener Universität. H. Obersteiner, Heft 2, Wien, 1894.

[4] Soc. de Biologie, April 14, 1894.

[5] J. J. Putnam: Relation of Infectious Processes to Diseases of the Nervous System. THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, March, 1895.

[6] Ueber diphtheritische Lähmungen. Virchow's Archiv, Bd. cxxiv. p. 2.

[7] Cramer: Aetiologie und pathologische Anatomie der enpheren Neuritis. Centralblatt für allg. Pathologie und path. Anatomie, Dec. 1893.

Thus we see that the pathological changes produced in the nervous system by diphtheria are not confined to a single definite variety of lesion. The cases of meningitis are probably due to secondary infection of the meninges, sometimes with the Klebs-Löffler bacillus, at other times with some of the other varieties of bacteria so often associated with this bacillus in the disease. The first, however, is not at all improbable, considering the frequency with which this bacillus has been found by Wright[6] in the various internal organs, and in the blood from the heart, in cases dying from diphtheria. Again, certain permanent paralyses,[7] occurring after diphtheria, as the rapid paralysis of one cranial nerve and the progressive ophthalmoplegias and paralyses of other cranial nerves, are probably nuclear in origin, the result of acute or chronic nuclear changes, sometimes inflammatory, at other times due to small hemorrhages, which we have so repeatedly found mentioned among the pathological changes.

The flaccid form of the ordinary diphtheritic paralyses, the condition of the electrical reactions in them, and the almost invariable recovery from them, all point to the lesion being one of the peripheral neuron. Whether the change is of the nature of a parenchymatous or an interstitial inflammation is harder to decide. We can, however, assume that the nutrition of the cell suffers from the presence of toxic products, probably chemical, in the blood, produced during the course of the disease, either by the micro-organism causing the disease, or by the body itself, acting under the stimulus of these micro-organisms or their products. As the axis-cylinder is but the prolongation of the nerve-cell, it is not surprising that changes are described as found in the cells of the cord at times, since the nerve-cell of the peripheral neuron in the cord may easily suffer with its process, though the changes are not those of an anterior poliomyelitis, as Dejerine thought. To be sure, certain facts are difficult to explain as resulting from a simple peripheral neuritis. Such as, for example, that the palate so often suffers alone, or that accommodation is affected without the involvement of other ocular muscles, while we should expect a toxic substance circulating in the blood to affect all nerves in approximately the same degree. To discuss these questions would take us too far from the subject of this paper, but the case of Kussmaul, quoted by Meyer,[1] is of sufficient interest to note here. In this case a newborn baby was attacked by diphtheria of the navel, and in consequence a paralysis of the abdominal muscles followed. This case seems to show that there is some influence from the proximity to the point of production of the poisonous substances produced during the course of the disease, perhaps upon the finer nerve-endings, which were indeed found affected in this case.

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[1] Gowers : Diseases of the Nervous System. 2d ed., vol. i. p. 112.

[2] Loc. cit., p. 235.

[3] Loc. cit., vol. ii. p. 913.

[4] Gazette des hôpitaux, 1877, p. 13.

[5] Archives de physiologie, 1880.

[6] J. H. Wright : Studies in the Pathology of Diphtheria. Boston Medical and Surgical Journal, Oct. 4 and 11, 1894.

[7] See Gowers : Diseases of the Nervous System, vol. ii. p. 909.

That cases of paralysis due to disease of the peripheral neuron, a peripheral neuritis, are not the only form of paralysis from diphtheria we have already seen in the case of the nuclear affections just mentioned. Besides these we find disease of the cord, and of the cord and brain combined, as in the cases of multiple sclerosis which have been reported.[2]

We find, in addition to these unusual forms of disease of the nervous system from diphtheria, still another form of paralysis. In the course of this disease we find a paralysis of the hemiplegic type, occurring suddenly, indicating a vascular lesion within the brain itself, caused by embolism, thrombosis, or hemorrhage. In the first cause we have to do with an embolus, carried from a clot formed in the heart, and lodging in one of the cerebral vessels. This is the direct result of the effect of the diphtheritic poison upon the muscle of the heart-wall, which has long been known and carefully studied. Leyden[3] found an interstitial myocarditis, and Mosler,[4] Oertel,[5] Rosenbach,[6] Birch-Hirschfeld,[7] Schemm,[8] and Unruh[9] have all described changes in the hearts of patients dying of diphtheria. Embolism from a heart-thrombus is known to occur in other marantic diseases, as typhoid fever, purpura, measles, scarlet fever, and epidemic cerebro-spinal meningitis.

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[1] Loc. cit., p. 214.

[2] A. Schönfeldt: Ueber multiple Hirn-Rückenmarksclerose nebst Angabe zweier Fälle bei Kindern nach Diphtherie. Inaug. Dissert., Berlin, 1888.

[3] Zeitschrift für klinische Medicin., Bd. iv. 1882.

[4] Archiv der Heilkunde, 1873, xiv.

[5] Deutsches Archiv für klinische Medicin, 1871, viii.

[6] Virchow's Archiv, Bd. lxx ; 352.

[7] Jarhresbericht der Gesellschaft für Natur. und Heilkunde, Dresden, 1879.

[8] Virchow's Archiv, Bd. cxxi. p. 235.

[9] Jahrbuch für Kinderheilkunde, Neue Folge, Bd. xx.

In the case of thrombosis in a cerebral vessel, which is not secondary to an embolus, we may have either a plugging of the vessels by bacteria in the blood-current, or the effect, direct or indirect, of the disease upon the vessel-walls, aided in most cases by the slowing of the blood-current, because of the affection of the heart.

Lastly, we have the cases of hemorrhage. The frequency with which, as we have seen, capillary hemorrhages have been found throughout the whole central nervous system points to a diapedesis of blood-corpuscles and an increased permeability of the vessel-walls, produced by the poison of the disease or by the direct action of the bacteria themselves, which we may infer both from the fact that they have been found within these small hemorrhages, and from the frequency with which they have been found in other organs of the body.[1] Auerbach[2] calls attention also to the increased blood-tension from the complicating nephritis so often occurring, as a condition favoring hemorrhage. Apolant[3] takes the ground that these small hemorrhages are the essential factor in the ordinary forms of paralysis from diphtheria, and that the hemiplegic form differs therefore from the other forms of paralysis quantitatively only, and not qualitatively. Certainly the hemorrhages large and small are produced directly by the poison of the disease within the body, and so should be considered as due to the disease itself. In like manner the changes in the peripheral nerves, which are the cause of the ordinary forms of diphtheritic paralysis, are produced directly by the disease, though not by the micro-organisms of the disease directly, as, indeed, in all probability, is the case with most of the symptoms of the disease. Therefore we are justified in speaking of diphtheritic paralysis and also of diphtheritic hemiplegia, as the element of time in the sequence of direct results of the disease, which constitutes the chief distinction in this case, is of minor importance.

Thus while the lesion producing these hemiplegias is the same as that in adult hemiplegias, yet they are so directly the result of the disease that it seems to me to be better to emphasize this difference by the use of a distinctive name. One writer, in reporting a case of this kind, calls it a cerebral palsy of childhood following diphtheria, although he does not discuss the pathological basis of the hemiplegia. I must dissent most decidedly from the use of such a name, because of the confusion which would result if such a designation should come into general use. In the large majority of the cases of the spastic cerebral hemiplegia of childhood we find the lesion in the cortex, most of them probably due to a meningeal hemorrhage, or an inflammatory process of the cortex, resulting in sclerosis, or the condition termed porencephalitis by Strümpell, and thus differing most decidedly from the cases we are now considering, which are usually due to a lesion of the internal capsule, except, perhaps, in the remarkable case of Gnauck,[1] where there undoubtedly existed a cerebral lesion of uncertain location, but very probably in the basal ganglia, indirectly involving the pyramidal fibres in the internal capsule for a time only.

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[1] Wright: loc. cit.

[2] Deutsche medicinische Wochenschrift, 1892, xviii. p. 168.

[3] Hemiplegie nach Diphtheritis. Inaug. Dissert., Berlin, 1894.

Cases of diphtheritic hemiplegia are rare, and most of the text-books upon nervous diseases, as well as those upon children's diseases and general medicine, are entirely silent upon the subject. Gowers[2] says: "In the course of this (diphtheria), as in most other acute specific diseases, sudden hemiplegia may indicate the occurrence of a vascular lesion of the brain, either thrombosis, embolism, or a hemorrhage. Death may result. The paralysis is often permanent." Von Ziemssen[3] says: "Nevertheless rare exceptional cases do occur, which offer difficulties in diagnosis, such as those which arise after slight preceding symptoms, with hemiplegias and monoplegias, ushered in by apoplectic attacks. Such cases are, however, in contradistinction to the typical picture of diphtheritic paralysis, the greatest rarities, and will scarcely come to the observation of even very experienced physicians." Henoch[4] mentions three cases in which he saw hemiplegia come on after diphtheria. One case ended in death, where the autopsy showed embolism of the artery of the fossa of Sylvius. Prof. Bókai, of the Stephanie Kinderhospital in Buda-Pest,[5] says that in this hospital since 1874 he has seen four cases of post-diphtheritic hemiplegia. Hirt,[6] in speaking of cerebral hemorrhage, says: "Exceptionally hemiplegia occurs after diphtheria, sometimes in conjunction with a paralysis of the palate, sometimes independently." Dillon Brown[7] says: "The paralysis of diphtheria may be divided into two classes: first, a true multiple neuritis, and, second, other types of paralysis as a result of hemorrhages or degenerative changes in the brain or spinal cord." Bernhardt[8] says that cerebral hemiplegia after diphtheria is very rare. Monti[9] says: "Generally the diphtheritic paralysis is symmetrical, only exceptionally does one observe the hemiplegic form." Eichhorst[10] says that rarely there occur in diphtheria hemiplegias of cerebral character, and that they are sometimes accompanied by aphasic disturbances. Sanné says:[11] "The paralysis is ordinarily symmetrical.

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[1] Centralblatt für Nervenheilkunde, 1891, p. 343.

[2] Loc. cit., vol. ii. p. 909.

[3] Klinische Vorträge. 6. Vortrag. iv. Nervensystem, 1. Ueber diphtheritische Lähmungen und deren Behandlung, p. 22.

[4] Vorlesungen über Kinderkrankheiten, 6te. Auflage, pp. 742, 751, 764.

[5] Orvosi Hetilop. No 24, quoted by Donath: Neurolog. Centralblatt, 1893, xii. p. 481.

[6] Diseases of the Nervous System. English translation of Hoch and Smith. Appleton, 1893, p. 219.

[7] In Louis Starr's American Text-Book of the Diseases of Children, 1893, p. 182.

[8] Berliner klinische Wochenschrift, 1886, p. 538.

[9] Eulenburg's Realencyclopädie, 2te. Auflage, Bd. v. p. 407.

[10] Handbuch der speciellen Pathologie und Therapie, 4te. Auflage, Bd. iv. p. 671.

[11] Traité de la diphthérie, Paris, 1877, p. 157.

It assumes very rarely the hemiplegic form. I have, for my part, met one case of right hemiplegia." Mendel[1] says that he has seen three cases. Wallenberg,[2] in 160 cases of hemiplegia, found 9 after measles, 13 after scarlet fever, and 6 after epidemic cerebro-spinal meningitis. Of Osler's 120 cases none followed diphtheria.

The reported cases of diphtheritic hemiplegia which I have been able to collect from the literature are twenty-eight in number.

Duchenne[3] mentions a case of diphtheria in which paralysis of the palate followed, where there came on suddenly in the middle of the convalescence, a diplegia lasting one hour, and thereafter a complete left-sided hemiplegia.

Henoch, at the Gesellschaft der Charite-Aertze in Berlin, at the meeting of February 11, 1886,[4] described one case which he had seen in a nine-and-one-half-year-old girl, who was taken with diphtheria on November 21st. On November 27th she was admitted to the hospital, and tracheotomy was performed. She had considerable albuminuria, which gradually diminished, with the improvement of the general condition. On the tenth day of the disease paralysis of the palate appeared. On the sixteenth day she was very weak, with a very slow pulse (52), which was small and irregular, and she had attacks of syncope. At this time there was very little albumin in the urine. She was unable to swallow and vomited. The respiration was 68 ; she had dyspnœa and tracheal râles. The pulse was 120. Beginning with the twenty-fifth day the respiration improved, until on the thirty-fourth day it was normal in frequency. The albumin was gone from the urine. On the forty-first day she had a sudden attack of paralysis of the left extremities and of the left side of the face, while the tongue deviated to the left. This condition lasted ten days. Then the hemiparesis disappeared gradually, and the improvement continued rapidly. At first the movements of the extremities were plainly ataxic. This also disappeared, as did the paralysis of the palate. The knee-jerks were absent on both sides. The patient recovered completely. Henoch thought the paralysis was due to embolic thrombosis.

Henoch then mentioned another previous case, where left-sided hemiplegia appeared in a nine-year-old girl after a severe attack of diphtheria. At the autopsy there was found a complete occlusion by thrombosis of the right Sylvian artery, without alterations having as yet appeared in the structure of the brain.

Henoch mentions a third case in his *Vorlesungen über Kinderkrankheiten.*[5] In this case, six weeks after the beginning of the diphtheria, when the patient was feeling quite well, there came on suddenly a right hemiplegia, which was not preceded by any paralysis of the palate.

In the discussion, at the meeting of the Gesellschaft der Charite-Aertze, which followed Henoch's report of these cases, Senator[6] mentioned, without giving details, that he had seen a case of well-marked left hemiparesis in a girl of fifteen years, who had had diphtheria at the age of four years. He states that the hemiparesis was cerebral, but without contractures, and with atrophy.

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[1] Zur Lehre von den diphtheritischen Lähmungen. Neurolog. Centralblatt, 1885, p. 133.

[2] Quoted by Sharples, Medical News, Philadelphia, Aug. 4, 1894.

[3] Bull. de Thér., lxxviii. fév. 28.

[4] Berliner klinische Wochenschrift, 1886, p. 538.

[5] 7te. Auflage, p. 759.

[6] Berliner klinische Wochenschrift, pp. 886, 1539.

Sanné[1] says: "I have for my part met one case of right hemiplegia," but gives no details.

Prof. Bókai, of Buda-Pest,[2] as has been said above, has seen four cases of diphtheritic hemiplegia since the year 1874. One case was in a boy of thirteen years, who had a complete right-sided hemiplegia, with involvement of the face and aphasia. In the other three cases there was once a right-sided paralysis of the face and aphasia, while in the second and third cases there was once a paralysis of the right and once of the left extremities. Bókai ascribed these cases to embolism of the artery of the fossa of Sylvius, due to a marasmic heart-thrombus.

Hirt[3] says: "In a girl, aged fifteen years, under my care, hemiplegia developed fourteen days after diphtheria, without any simultaneous disturbance of consciousness, and only slight improvement was noticed after several years."

Mendel[4] states that he has seen three cases of hemiplegia after diphtheria, two of which died. One case lived, but remained hemiplegic, and contractures developed. In all three cases there was a hemiplegia involving the lower portion of the face, the arm, and the leg, without disturbance of sensation, twice on the right and once on the left side. It came on suddenly, without marked disturbance of consciousness. One case came to autopsy, when there was found a hemorrhage the size of a cherry-stone into the inner portion of the lenticular nucleus, which had destroyed a part of the neighboring inner capsule.

In the discussion of this paper Prof. Remak[5] mentioned two cases of the same affection.

One was the case of a seven-year-old girl, who had an attack of loss of consciousness, followed by right hemiplegia, with transitory aphasia. The other case was one of left-sided spastic hemiplegia in a boy of eight years.

Solbrig[6] reports another case. E. E., four years of age, was admitted to the hospital on May 15, 1884. She had been ill with diphtheria from the 14th of April until the 1st of May. Three weeks previous to admission she had been attacked by right-sided paralysis without loss of consciousness or convulsions. At the time of admission there was paralysis of the lower portion of the right side of the face, the head was bent to the left, and the chin turned toward the right. There was also paralysis of the right upper and right lower extremities, which could not be moved at all. There was paralysis of accommodation and of the palate. The knee-jerks were absent. There was aphasia. Sensation was not affected. On June 10th the patient could speak, and there was right convergent strabismus. On August 5th the paralysis of the face was gone. The extremities were still affected. There was paralysis of the thumb-muscles and of the right peronei. The remainder of the muscles of the right extremities were still paretic. The speech was normal. The knee-jerk was absent on both sides. The paralysis of the palate, of accommodation, and the strabismus were gone. On May 5,

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[1] Loc. cit., p. 157.

[2] Loc. cit.

[3] Loc. cit., p. 219.

[4] Loc. cit.

[5] Berliner klinische Wochenschrift, 1885, No. 13, p. 203.

[6] Otto Solbrig: Ueber nervöse Störungen nach Diphtherie. Inaug. Dissert., Berlin, 1886.

1885, there was still some paralysis in the region of the facial nerve, and the uvula was drawn down to the right side. Speech was normal. Contractures had formed in the muscles of the forearm. Active motion was retained in part—almost completely in the right foot. The knee-jerk on the right was increased. There were contractures in the muscles of the right leg, so that pes equinus had resulted.

Bouchut[1] reports a case of diphtheria where suddenly, during the night, right hemiplegia came on. There was paralysis of the palate, a "congestive neuritis" in the right eye, embolism of the superior branch of the central artery, and in the left eye optic atrophy. The heart was normal. The author seems uncertain as to the cause, but certainly embolism seems the most probable one.

Another case of hemiplegia from diphtheria is reported by Jäckle.[2]

G. A., five and one-half years old, was taken ill in March, 1891, with diphtheria. He became free from fever on the fifth day. On the eighth day the membrane was gone. The heart and lungs were normal. On the ninth day œdema of the face was noticed, and the next day of the legs. At that time there was albumin in the urine. The patient was apathetic, and vomited at times. The speech was nasal, but there was no difficulty in swallowing. On the twelfth day the patient had clonic and tonic convulsions, which were repeated several times. There was loss of consciousness. The feces and urine were retained. The pulse was 112, and hard in character. On the fourteenth day there had been no more convulsions, and the mind was clear, but there was now paralysis of the right side of the body, and the mouth was drawn to the left. The pupils reacted slowly, but were equal. Sensation was not disturbed. There was complete aphasia. Both knee-jerks were weak. The urine and feces were retained. The urine, which was drawn by a catheter, contained much albumin, epithelial casts, and epithelium. The condition remained practically the same until the end of the fourth week. Then the urine still contained a trace of albumin. The speech was better. The knee-jerks were normal. The paralysis was still unchanged. The faradic excitability of the nerves of the right side was somewhat diminished. At the beginning of the eighth week the ataxia and Romberg's symptom were still present, yet the patient could take a few steps. In the twelfth week the gait was better, though the right leg was still dragged. The arm had not improved so rapidly, but it could then be raised to the horizontal and feebly flexed at the elbow. The hand was held clinched and flexed at the wrist. The lower extremity became gradually quite capable of doing its work again, but motion of the arm remained impaired. The corner of the mouth was drawn somewhat to the left. The speech was normal. The author concludes that the cause of the hemiplegia was a hemorrhage into the posterior limb of the inner capsule.

Auerbach[3] reports a case of diphtheritic hemiplegia from the hospital at Frankfort-on-the-Main.

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[1] Hemiplégie diphthéritique droite. Cérébroscopie. Atrophie papillaire et embolie dans l'œil gauche; névrite congestive de la papille droite. Gaz. des hôpitaux, Paris, 1869, xlii. p. 401.

[2] A. Jäckle: Hemiplegia cerebralis nach Diphtheritis. Aerztliche Mittheilungen aus und für Baden, Mai 13, 1891, 1892, xlvi. p. 75.

[3] Ueber einen Fall von Hemiplegia cerebralis nach Diphtheria. Deutsche medicinische Wochenschrift, 1892, xviii. p. 168.

A. B., seven years old, was taken severely ill with diphtheria. The membrane was gone on the seventh day. On the eighth day there was paralysis of the palate, with nasal speech. There was no anæsthesia of the pharynx, and no paralysis of the constrictors of the pharynx. The urine was normal. On the twelfth day the patient became apathetic and vomited. The urine was cloudy; the amount small (300 c.c.); albumin was present; and many casts were found, which were granular and epithelial. On the fourteenth day œdema of the face was noted. The urine had not changed, but there was less vomiting. The examination of the chest was negative. The paralysis of the palate continued, but the muscles of deglutition remained unaffected. On the twentieth day of the disease, and eight days after the appearance of the nephritis, there came on tonic and clonic spasms of the whole body, of ten minutes' duration. The pulse was 120, and of high tension. The temperature was normal. The patient was unconscious, and this lasted throughout the night. There was no dyspnœa or vomiting. There was almost complete anuria. The mind of the patient was somewhat affected the next day. The muscles of the eyes, including that of accommodation, were not affected. The right corner of the mouth was drawn to the right, while the left cheek and the left side of the chin were motionless. The forehead was not affected. The uvula was drawn somewhat to the right. There was complete motor paralysis of the whole of the left side of the body, while the right side was not affected. Sensation on the left side was normal. There was absence of the knee-jerk and of the abdominal reflex on both sides. There was no difficulty in the speech like aphasia. There was no incontinence of urine or feces. The urine still contained albumin. There was no vomiting. On the twenty-third day there appeared moderate ascites, double hydrothorax, and anasarca. The paralysis remained unaltered. On the twenty-fifth day the œdema and the amount of albumin in the urine had diminished, while the amount of urine had increased. On the twenty-eighth day there was no albumin and no œdema, and the reflexes were normal. The faradic excitability of the nerves of the left arm was a little diminished, otherwise there was nothing abnormal in the electrical reactions. The patient improved from the fourth week. By the beginning of the seventh week she could raise the left leg half as high from the bed as the other leg. One week later she made her first attempt at walking, on which the left leg was dragged. There was then well-marked ataxia, and Romberg's symptom was present. At the time of the patient's discharge the left leg was still dragged in walking, the strength in the left hand was markedly less than in the right, and it was easier to straighten the left arm from the flexed position than the right. The arm could not be flexed and only slightly extended. The fingers could be flexed, or spread, only slightly. There existed some disturbance of co-ordination in the left arm. There was no atrophy. In laughing the mouth was drawn to the right. The organs of sense were normal. The speech was slightly nasal. The intelligence was normal. Three weeks later there was slight atrophy in the left arm. Auerbach thought he had to do with a hemorrhage in the anterior two-thirds of the posterior limb of the inner capsule, and in connection with this an ordinary diphtheritic paralysis. He concludes that it could not have been a case of uræmic hemiplegia, as these are known to be transitory.

Prof. Edgren, of Stockholm, reports another case.[1]

O. L., ten years of age, was taken ill on November 15, 1890, with diphtheria. He was treated in the hospital for one month. During this time he had severe vomiting. About three weeks after the attack he suddenly fell from his seat. Whether he was unconscious or not is not known, nor whether the paralysis came on at once or not. The next day it was noticed that there was paralysis of the right side, with aphasia. The patient remembered that he had had for a short time twitching of the right arm. The patient was taken home on December 17th. He was then very weak, the right corner of the mouth was lower than the left, the right cheek and chin were motionless. The eye-muscles were normal. The right arm and leg could be moved feebly. Except for a few incomprehensible nasal sounds, speech was lost. Fluids came through the nose upon drinking. There was anæsthesia of the right side, which was also colder than the left. There was incontinence of urine and feces. The patient soon became better. At the end of January, 1891, he could walk with help. In February he was able to go out. Speech could be understood, but it remained nasal for many months. The drawing of the mouth to one side gradually lessened, the power of motion returned in the right arm, and the disturbance of the bladder and rectum disappeared. During the spring there was paresis of the accommodation, which was still present in the fall. On January 20, 1892, examination showed the patient of normal size, somewhat thin, and the muscles weak. The urine, the heart, and the lungs were normal. There was a very slight paresis of the lower portion of the right face. The soft palate and uvula moved alike on both sides. The tongue deviated to the left. The patient could walk and run. There was some stiffness of the right leg in walking, the toe was dragged a little, and the foot circumducted at each step. The right extremities were colder than the left. The right hand became blue upon hanging down. There was no atrophy. The reflexes at the elbow in the arms were equal. The knee-jerks were lively, the right more so than the left. Ankle-clonus was present on the right, and the Achilles tendon-jerk was greater on the right than on the left. The cremasteric and abdominal reflexes were equal upon the two sides. Sensation was good on both sides, except that the localizing sense was diminished upon the backs of the fingers of the right hand. The electrical sensibility and excitability were somewhat diminished on the right side, but there were no qualitative changes. At the time of discharge, on March 15th, the patient was almost entirely well. Edgren regarded this as a cerebral case, due to a hemorrhage, which probably affected the inner capsule of the left side. There was no cause for embolism.

Dr. Jones[2] reports a case of diphtheria in a girl, which lasted ten days, in the course of which, or after which—the author does not state the time exactly—a sudden right hemiplegia came on, with involvement of the right side of the face, and ataxic aphasia. There was no loss of consciousness. The sensation was normal. The knee-jerks were weak and equal. There was no pain. The palate was paralyzed. There were no heart-murmurs. Two days later the patient began to move the leg, and motion returned gradually, so that in six weeks she was entirely well.

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[1] J. G. Edgren: Ueber einen Fall von halbseitiger Lähmung nach Diphtheria. Deutsche medicinische Wochenschrift, 1893, xix. p. 864.

[2] A. A. Jones: Medical News, Philadelphia, Oct. 22, 1892, p. 417.

Seifert[1] reports two cases. The first was in a girl of ten years, rather weak, not hereditarily affected, but not very well developed mentally. She had always been well. The diphtheritic membrane had disappeared on the eighth day, and there was no more fever. On the tenth day there appeared paralysis of the palate, with nasal voice, and regurgitation of food through the nose. The palate was not moved on phonation. The action of the heart was disturbed, the sounds being quite irregular in rhythm and strength, and later they became very weak and intermittent. The heart was found enlarged, by percussion, as far as to the middle of the sternum. There were no murmurs. Respiration was moderately quickened. The pulse was small, easily compressible, and intermittent. There were no symptoms of kidney-trouble, and there was no albumin in the urine. On the eleventh day the heart-weakness increased, and there were symptoms of collapse. In the following night the patient awoke suddenly with a cry, stared about her, and seemed confused. She was found to have a right hemiplegia. There were no convulsions or twitchings. The right side of the face was affected, except the upper portion. The tongue deviated to the right. The right upper and lower extremities were motionless. The abdominal reflex was absent. There was ataxic aphasia. The patient understood what was said to her, but could not speak. Later she said, " Anne, Anne," to everything. Sensation was normal. The muscles of the eyes, the pupils, and the whole left side were normal. For the next few weeks there was no change in the paralysis or in the condition of the heart. Then there developed a paralysis of deglutition and an anæsthesia of the pharynx. The patient was in great danger till the end of the sixth week. In the seventh week she could swallow better, and the general condition had improved. The power of speech returned gradually, and also motion, first in the leg and later in the arm. By the ninth week she could attempt to walk. During the following weeks motion increased, but there now appeared spasm in the muscles and increased tendon-reflexes on the right. The condition six months later was this: the right side of the face was still paretic, the tongue was protruded straight, the speech was normal, the right arm could be raised vertically, and the elbow moved freely. The hand and fingers were held flexed by contractures, and extension, abduction, and adduction of the wrist and fingers were much limited, but flexion less so. There was slight weakness in the extensors of the right lower leg. The knee-jerk was increased. No clonus. Slight spasm in the leg and more marked spasm in the arm. Sensation was normal, as it had been all along. Seifert regarded embolism as the cause of this hemiplegia, the weakness of the heart having given rise to a thrombus there, which was the cause of the embolism.

The second case which Seifert reports[2] was that of a girl of nine years, who was attacked severely by diphtheria. On the seventh day there was paralysis of the palate. From the fourteenth day there were nephritis, œdema of the face and legs, scanty, cloudy urine, which contained albumin, and numerous granular and epithelial casts. On the twentieth day there were severe headache, vomiting, and apathy. On the next day sudden and complete hemiplegia came on. The patient cried out several times, became completely unconscious, and this condition lasted the whole day. There were no convulsions. The tongue deviated to the right, and the right side of the face and the right upper and lower extremities were relaxed and paralyzed. The knee-jerk and the abdominal reflex on the right side were absent. There was no disturbance of sensation. The muscles of the eyes and the pupil-reactions were normal. When the patient became conscious, she was found to have complete motor aphasia. She showed by signs that she understood everything, but was unable to speak. The heart and lungs were negative. There was some headache, but no dizziness or vomiting. The condition of the kidneys improved. In the course of the next four weeks there was a return of speech, and of movement in the right leg and later in the right arm. The recovery was slow. It was five months before she was able to walk, and then the right leg was dragged. She could not stand without help. There was limitation of motion in the right shoulder and elbow-joints, and the hand and fingers could not be moved at all, but were held flexed by contractures. There was marked spasm of all the muscles of the paralyzed limbs. The tendon-reflexes were increased. On March 27, 1893, the following condition was found: There was slight weakness in the muscles supplied by the lower branches of the right facial nerve. The tongue did not deviate. Speech was normal. The right arm could be raised vertically, flexion and extension at the elbow were possible. There was only slight motion in the hand and fingers. The contractures in the fingers could be overcome only with difficulty. The right leg was dragged a little in walking, and the gait was paralytic. The right knee-jerk was increased. There was no ankle-clonus. There was a spastic condition of all the muscles of the right leg. Motion at the ankle was limited. The electrical irritability in the paralyzed limbs was slightly diminished, but not changed qualitatively. There were no trophic disturbances, and no anomalies of sensation or co-ordination. In this case Seifert concludes that since the heart was negative, the hemiplegia was due to a hemorrhage which had affected the inner capsule of the left hemisphere.

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[1] P. Seifert: Ueber Zwei Fälle von Hemiplegie cerebralis nach Diphtherie. Neurologisches Centralblatt, 1893, xii. p. 113.

[2] Loc. cit.

Another case has been reported by Donath.[1] An eight-year-old peasant boy was taken ill on November 22, 1892, with diphtheria, which lasted fourteen days. On the third day of convalescence he began to go about. There came on that night while he was asleep a complete hemiplegia, with marked involvement of the face, and aphasia, which was complete for fourteen days. The mother said that he could not speak a single word, but understood everything that was said to him. The paralysis of the face improved in three weeks, the aphasia in four weeks. When he began to speak again he was hoarse, probably from a paralysis of the vocal cords. He had no difficulty in swallowing. During the next two months there was no change. The condition on April 22, 1893, was that of a well-developed, moderately nourished, but somewhat anæmic boy, with paralysis of the lower branches of the right facial nerve, and moderately indistinct and drawling speech. The palate could be moved perfectly. The right extremities, especially the upper extremity, were paretic. The upper extremity was held flexed by moderate contractures. The body was held leaning toward the right. The right leg was dragged in walking. The right triceps and patellar tendon reflexes were much increased, and there was also ankle-clonus. On the left side all was normal. The sensations of touch and pain were tolerably prompt on the right, but somewhat less plain than on the left. The right upper extremity was slightly diminished in volume, the right lower extremity less so. There was nothing remarkable about the electrical reactions. There was no paralysis of accommodation. The right eye had 7.0 D. of hypermetropia, and there was convergent strabismus. In the left eye there was slight myopia. On May 18th the speech was still indistinct. The face was nearly normal. The extremities were more useful. The arm could be raised and stretched out. The patient could run moderately well. Donath concludes that since five months had elapsed and contractions had formed, the paralysis was due to hemorrhage.

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[1] J. Donath: Ein Fall von diphtheritische Hemiplegie. Neurologisches Centralblatt, 1893, xii. p. 481.

Sharples[1] reports another case in a boy of thirteen years. There were cases of diphtheria and erysipelas in the house, and the boy had diphtheria. Before the membrane was gone the heart became very rapid and irregular. At midnight, December 4, 1893, he became suddenly paralyzed on the right side. There were no convulsions. He cried out several times during the evening preceding the attack. Twenty-four hours later there were tremors on the right side which shook the whole body. The skin was extremely hypersensitive. The tendon-reflexes were absent. There was aphasia. The heart was irregular and weak, but there were no murmurs. The urine was scanty, but it is not mentioned whether albumin was present or not. Four months later the gait was hemiplegic. Motion was better, but there was paresis on the right side. There was no athetosis. The tendon-reflexes were increased, but there was no ankle-clonus. The skin-reflexes were increased. There was no reaction of degeneration in the muscles of the right side. There was moderate atrophy of the right upper and lower extremities. The aphasia was improving. Sharples thinks the hemiplegia in this case was due to embolism.

Apolant reports another case.[2]

G. R., thirteen and one-half years old, had had measles, chickenpox, and scarlet fever, with nephritis. At the beginning of April, 1892, he had a severe attack of diphtheria. The mother noticed that fluids regurgitated through the nose, and that the voice was nasal. There were at this time œdema of the face and a small amount of concentrated, cloudy urine. During the night of the 15th–16th of April the patient was very restless and cried out. The next morning he was conscious, but remembered nothing about the night. The mother then noticed that the arm hung flaccid by the side, and that he walked with difficulty. The weakness of the right arm and leg is said to have increased gradually. In June he went to school again, but could not write, as he had twitching of the fingers of the right hand, and he could not see well (paralysis of accommodation?). He often fell when running. Walking was also difficult, and the right arm could not be used well. He was said to have had no disturbance of speech, except that it was nasal. The difficulty in moving the arm and leg continually increased, and sensation was also diminished on the right. On June 20, 1894, the patient came to Prof. Mendel's clinic, where it was noticed for the first time that the right lower facial muscles were paretic, and that the tongue deviated to the right. The uvula was in the middle. The pupils were equal and reacted to light. The external ocular muscles were normal. The heart was negative. There was paresis of the right arm with weakness and spasm of the muscles. No disturbance of micturition. The right leg was weaker than the left, and there was especial difficulty in moving the toes. The patellar reflex was greater on the right than on the left. Sensation was diminished over the whole right side. Examination on July 3, 1894, showed the patient to be of normal size, but poorly developed, and with small muscles and little fat. The skin and mucous membranes were pale. The heart was negative. The urine was normal and contained no albumin. The muscles of the right side of the face were more relaxed than those of the left side, and that side hung down a little more and moved less than the left side. The upper part of the face was not affected. The pupils reacted normally to light and upon accommodation. The tongue deviated slightly to the right, and the uvula was drawn slightly to the left. The left side of the soft palate moved better than the right upon phonation. The speech was normal, but gave the impression that it required more effort than normally. The right shoulder could be elevated much less than the left. The right arm hung relaxed, the elbows slightly bent, the hand midway between pronation and supination, the fingers straight. The arm could be raised almost to the vertical, but less easily than the left. Passive motion in the shoulder was very slightly difficult, in the elbow more so, because of a slight spastic contracture of the biceps and supinator longus muscles. The wrist-joint was freely movable, the fingers less so. Active flexion of the fingers was particularly difficult, and somewhat so were also their extension, abduction, and adduction. There was no evident atrophy of the right arm. The grasp on the right was fair, but weaker than on the left. The patient could walk well, but the gait was paretic and spastic. The right leg, which was moreover plainly atrophied in the thigh, was held stiffer at the knee-joint than the left, and was swung outward in walking. Motion at the hip and knee was not so easy on the right as on the left. Flexion of the right leg at the knee was plainly weaker than that of the left. The ankle-joint was freely movable. The toes, except the great toe, could hardly be moved at all. The great toe lay over the second toe. There was slight contracture of the plantar flexors, especially the flexor digitorum communis. In regard to sensation, the patient said he felt more plainly on the left than on the right. Temperature-sense, muscle-sense, and locality-sense were normal. The knee-jerks were lively, the right the greater. The abdominal and cremasteric reflexes were normal. There was no ankle-clonus. The electrical reactions showed no abnormality. There was no ataxia, and Romberg's symptom was absent. Apolant concludes that the lesion was situated in the internal capsule of the left hemisphere, but does not attempt to decide whether it was produced by a hemorrhage or an embolus.

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[1] C. W. Sharples: Cerebral Palsy of Childhood Following Diphtheria. Medical News, Philadelphia, August 4, 1894, p. 124.

[2] E. Apolant: Hemiplegie nach Diphtheritis. Inaug. Dissert. Berlin, 1894.

In the Berlin Gesellschaft für Psychiatrie und Nervenkrankheiten, Gnauck[1] presented another case of diphtheritic hemiplegia. A nine-year-old boy in his fourth year had diphtheria severely. Fourteen days later there was paralysis of the left side, with involvement of the left side of the mouth. After five weeks the paralysis disappeared. In the sixth week there came on a new attack, with vomiting, pain in the left side, and delirium. The condition of the patient when shown was that there were athetoid movements in the left hand. These movements also involved the toes, and the left side of the face as well. The shoulder was shrugged often. There was diminution of sensation on the left side. Gnauck thought that the patient had a central affection after diphtheria.

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[1] Centralblatt für Nervenheilkunde, 1891, p. 343.

To these twenty-eight cases of diphtheritic hemiplegia I am able to add two cases: one seen at the Boston City Hospital in the service of Dr. Knapp, and the other seen at the Boston Children's Hospital in the service of Dr. Bullard, to both of whom I wish here to express my thanks for their kind permission to make use of the cases.

The first case was that of Miss A. J., who came to the Boston City Hospital on April 23, 1894, She was twenty years of age, and single. Her family history was good, and she herself had always been well. When she was twelve years old she had what appeared to be an attack of diphtheria, for which she was treated at home. The trouble in the throat did not last long, how long could not be determined exactly, and the patient was soon convalescent. During convalescence paralysis of the palate came on, and fluids were regurgitated through the nose on swallowing. The sight was not affected at that time. Once during the convalescence she lost consciousness, but she had no convulsions. Whether this was due to cardiac weakness, or was the effect of the lesion of the brain which produced the later trouble, it is impossible to say. Only when she was able to be about again was it noticed that she could not use the right arm and leg as before her illness. The face was not affected so far as the patient knew. For some time she could not make herself understood when talking, though she understood everything that was said to her, but this trouble gradually disappeared in time. She had no pain in the limbs or head. At no time during the illness were the feet swollen, and there were no other signs of kidney-trouble noticed. The ability to use the right arm and leg increased, but she always had a considerable amount of difficulty particularly in the use of the hand. Physical examination showed a well-developed and nourished girl, who was slightly anæmic. She limped slightly with the right leg in walking, and the toes of that foot were not lifted well, but they were not dragged, and the leg was not circumducted, nor was the gait distinctly paretic or spastic. She did not sway when standing with the eyes closed. The right arm was usually held by the side, the elbow extended, the forearm supinated, the fingers flexed, the wrist not flexed but a little abducted. The right foot showed moderate pes equinus. There was considerable spasm of the muscles of the right lower extremity, and a general paresis which was shown most clearly in the extensors of the right foot. The rigidity of the right upper extremity was less, but was present, and the right arm was distinctly weaker than the left, which was shown chiefly in the grasp, which was decidedly weaker than that of the left hand, but also in flexion of the forearm on the arm, and in pronation and supination of the forearm. The rigidity of the arm was increased upon movements of the leg, but there was no athetosis. The right thigh measured 41.5 cm., 20 cm.

above the upper border of the patella. The left thigh, at the same point, measured 44 cm. The circumference of the right calf was 30 cm. That of the left calf, 37 cm. The left arm measured 25 cm., the right the same. The left forearm was 22 cm. in circumference, and the right 23 cm. The triceps tendon reflexes in the arms were equal. The knee-jerks were equal and lively. There was no ankle-clonus. There was considerable difficulty in using the right extremities, chiefly on account of the spastic condition, less because of the paresis. There was perhaps a very slight disturbance of co-ordination in both the arm and the leg. There was no disturbance of the muscular sense nor of the sense of position. Sensation was everywhere normal. There was no paresis of the facial muscles. The tongue was protruded straight. The two sides of the soft palate moved equally on phonation. The pupils were equal, and reacted to light and with accommodation, as well as to pain, and the associated action was also normal. There was no affection of the external ocular muscles. Speech was normal. The anterior tibial group of muscles in the right leg required a strong faradic current in order to produce any reaction. The other muscles of the right side reacted normally. There were no qualitative changes in the reactions to the galvanic current. The area of cardiac dulness was not enlarged. There was a soft systolic murmur over the pulmonic area. The second pulmonic sound was not accentuated.

The second case was that of a boy, P. G., twelve years of age, who came to the Boston Children's Hospital on May 23, 1894. He was the oldest of seven children, all of whom were living and healthy. He had had measles at six years, scarlatina at seven years, and pertussis at eight years of age. In October, 1893, the patient had diphtheria. He was ill about two months in all. About two weeks after the illness began the throat had improved so that he was able to be about the house again. About this time fluids began to regurgitate through the nose, and he could not see as well as before (probably from paralysis of the accommodation). He also said that he had diplopia. In November, some three weeks after the illness first began, he awoke from sleep one night with pain in the left extremities, which was worse in the arm, and found that he could not move either left extremity, and that the face was drawn to one side. The patient said that he was unable to speak at all. This difficulty lasted for about two months, by which time he was able to speak well again. About a week after speech was recovered he began to be able to walk again. He was admitted to the Boston Children's Hospital on May 12, 1894, about seven months after he was first taken ill. At that time there was marked atrophy (?) of the muscles of the left arm. The fingers were kept flexed, and he could not extend them. He was unable to pronate the hand. There was marked weakness of the grasp on the left side, and all the movements of the muscles of the left arm were much weakened. The shoulder-muscles on the left were also much weaker than on the right. The thigh- and leg-muscles of the left side showed muscular weakness in all directions. Sensation was not much affected. The knee-jerk on the left was distinctly greater than on the right. There was no ankle-clonus. Examination on May 23, 1894, showed a well-developed and nourished boy of normal size. There was paresis of the muscles supplied by the lower portion of the left facial nerve. The upper portion was not affected, and the left eye could be closed as firmly as the right. The tongue deviated to the left. The palate moved normally on phonation, and speech was not affected. There was paresis of the left arm, which was especially marked in the extensors of the wrist. There was considerable spasm of the muscles of the left arm on passive motion, and there was also slight atrophy of these muscles. Sensation was slightly diminished in the left arm, but less so in the lower extremity. There was slight paresis of the left lower extremity. The gait was normal, and Romberg's symptom was absent. The knee-jerk on the left side was greater than upon the right, but there was no ankle-clonus. The tendon-reflexes in the left arm were increased. The pupils reacted normally to light and with accommodation. There was no paralysis of the external ocular muscles, and no diplopia. There was slight inco-ordination in the left upper extremity, but no disturbance of the muscular sense or of the sense of position. There was no inco-ordination, disturbance of the muscular sense, or of the sense of position in the corresponding lower extremity. The electrical reactions showed a diminished excitability of all the muscles of the left side to the faradic current, but no qualitative changes to the galvanic current. The heart was negative.

Both these cases were evidently due to some vascular disturbance in the brain, and undoubtedly of the internal capsule of the left hemisphere in the first case, and of the right hemisphere in the second one. It was, unfortunately, impossible in either case to get a very accurate account of the symptoms at the time of the occurrence of the hemiplegia, and so it is impossible to say definitely in either case whether it was due to hemorrhage or embolism. We can only conclude in both cases, from the existence of permanent paresis and of muscular spasm, that the vascular lesion, whatever it was, had caused a destruction of nervous structures, which was followed by a secondary degeneration. As it was impossible to learn the condition of the heart or the kidneys at the time of the attack in either case, any help which might be obtained from knowing these facts is also excluded. It is of interest to note the fact that in the second case the patient could not speak for nearly two months after the hemiplegia came on. This rests upon his own unsupported statement, and could not be confirmed, as none of the relatives were seen, but there seems to be no reason to doubt the statement, as the boy told a straightforward story in other respects. However, the occurrence of aphasia with a left hemiplegia is certainly rare, but the fact that he could not speak at all, and that regurgitation of fluids came on previously without loss of speech, would indicate that there was a real motor aphasia in this case.

Of the thirty cases given more or less fully here, we find that the probable diagnosis in seven is hemorrhage, namely, one each of Mendel, Jäckle, Auerbach, Edgren, Seifert, Donath, and Solbrig. The embolic cases are ten, those of Buchut, Seifert, Sharples, Jones, two of Henoch's, and all four of Bókai's. The remainder, in which the diagnosis must remain more doubtful because of the ambiguity of the case or the too brief description, comprises one case each of Duchenne, Henoch, Senator, *Hirt, Apolant, and Gnauck, and two each of Mendel and Remak, and* the two added in this paper.

The attack ended in death in three cases, in two of which autopsies were made. These resulted in the finding of a thrombosis of the artery of the fissure of Sylvius in the case of Henoch, and a hemorrhage into the internal capsule in that of Mendel. Unfortunately, we have no cases in which bacteriological examinations have been made or the walls of the vessels carefully examined. In two cases, one of Henoch and one of Jones, recovery took place. In five other cases it is difficult to say whether the paralysis was permanent or not, namely, in those of Henoch, Sanné, Buchut, and the two of Remak, while that of Edgren was stated to be almost well at the time of discharge from the hospital. In the remaining nineteen cases the hemiplegia was permanent, in the sense that it caused a greater or less permanent disability.

Since this article was written, still another case of diphtheritic hemiplegia has been reported. This patient was shown by Dr. Jacobsohn at the meeting on July 8, 1895, of the Berlin Gesellschaft für Psychiatrie und Nervenkrankheiten (*Neurologisches Centralblatt,* 1895, xiv. p. 794). The eight-year-old child had had diphtheria about four months before. Five weeks after the beginning of the disease a nephritis developed, which lasted three weeks. During this time a right hemiplegia came on suddenly, without loss of consciousness or convulsions. The lower part of the face was affected as in ordinary cases of hemiplegia. The right shoulder was raised, and the right arm held pressed against the side, the upper arm generally rotated outward and drawn backward, the forearm overextended and pronated, the hand abducted toward the ulnar side, the fingers flexed, and the thumb flexed and adducted toward the index-finger. The right scapula at the upper part was nearer the median line than the left one, and it was also raised. The right arm could not be raised quite as high as the left, nor could it be held so long elevated. The right shoulder was raised less well than the left. Flexion and extension of the forearm were possible, as also pronation, while supination was very incomplete, even with the aid of the biceps. Extension of the hand and fingers was impossible. Flexion of these was normal. Flexion and adduction of the thumb was good, while extension and abduction were impossible. Sensation was normal. The knee-jerk was absent on both sides. There was no atrophy, no reaction of degeneration, and no tenderness to pressure of the peripheral nerves.

Goldscheider, in the discussion that followed, said he thought the cases of hemiplegia after diphtheria were not so rare as is generally supposed, and stated that he himself had seen several, which he had not published. This statement was modified later, Goldscheider (*Neurologisches Centralblatt,* 1895, xiv. p. 848) then stating that he had seen not several, but one case of hemiplegia after diphtheria.